new opportunistic infection, was first described when small, apparently gram-negative bacilli were noted on electron microscopic examination of a cutaneous lesion suspected of being Kaposi's sarcoma.

Cutaneous bacillary angiomatosis, sometimes called epithelioid angiomatosis, is marked by red papules and nodules and, less often, rounded subcutaneous masses. On a biopsy specimen, there is a proliferation of capillaries with greatly protuberant endothelial cells. The lesions may easily be mistaken for pyogenic granulomas, Kaposi's sarcoma, or angiosarcoma. Features that enable a specific diagnosis of bacillary angiomatosis to be made include numerous neutrophils, nuclear dust, and purplish granular material that proves, on electron microscopy or Warthin-Starry stain, to consist of bacteria. Most patients respond well to a regimen of erythromycin, 2 grams per day.

Recent attention has focused on the identity of the causative bacillus and on the spectrum of internal disease produced by the organism. Because the agent of bacillary angiomatosis causes vascular proliferation, there has been speculation that it is related to *Bartonella bacilliformis*, which causes an acute febrile illness known as Oroya fever and a chronic illness with numerous cutaneous vascular lesions known as verruga peruana. Recent studies using whole cell fatty acid chromatography have shown that the agent of bacillary angiomatosis has a similar profile to *Bartonella* species. Because some of the original patients had been scratched by cats and because the bacillus shows the same restricted staining pattern as does the bacillus of cat-scratch disease, the proposal has been made that bacillary angiomatosis is caused by the cat-scratch disease bacillus. This remains unproved. So far, culturing the bacillus using conditions favoring growth of the cat-scratch disease bacillus has proved difficult. A third possibility has been established by molecular biologic studies. Sequencing of the 16S RNA gene has shown that the agent of bacillary angiomatosis is most similar to a rickettsia, *Rochalimaea quintana*, the agent of trench fever, which is clinically unlike bacillary angiomatosis.

The spectrum of internal disease caused by this organism, with or without cutaneous lesions, has expanded. Soft tissue masses, lymphadenopathy, and dramatic hepatomegaly and splenomegaly have been seen in affected patients. Peliosis hepatis (the formation of blood-filled cysts in the liver) is an unusual consequence of infection. Clusters of bacilli inhabit the connective tissue that rims these cysts. The internal manifestations of bacillary angiomatosis also respond to antibiotic therapy. Fatal untreated cases have occurred.

The next few years will doubtless see more precise identification of the organism, as well as the documentation of additional cases in non–HIV-infected patients.

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Suntan Parlors—A New Hazard to Health

WITH THE DEVELOPMENT during the past 20 years of high-intensity lamps emitting UVA, the suntan facility industry has burgeoned. In California, it is estimated that there are about 2,000 such facilities.

UVA is long-wave ultraviolet light in the range of 320 to 400 nm. Unlike UVB (280 to 320 nm), it passes through window glass. It is present throughout the day and during the entire year. UVA penetrates the epidermis into the dermis. Because of this, it can damage not only the Langerhans cells in the basal layer of the epidermis but also collagen tissue, the lens and retina of the eye, and even blood vessels. A growing body of literature indicates the potential for the development of basal and squamous cell carcinomas, as well as the development of cutaneous melanomas on the basis of exposure to UVA.

Beginning in the 1980s, it was noted that an increasing number of eye injuries were caused by exposure to UVA in suntan facilities. Severe burning of the skin, exacerbation of autoimmune illnesses such as lupus erythematosus, photoallergic reactions of the skin, premature aging of the skin, an increase in the number of skin cancers, and one death have been reported. The use of suntan parlors apparently contributed to an epidemic of phytophotodermatitis among grocery workers handling fresh produce and fresh flowers.

It is hoped that an awareness of the hazards of UVA will lead to decreased exposure to this harmful agent.

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Carbon Dioxide Laser Treatment of Actinic Cheilitis

ACTINIC CHEILITIS is a diffuse premalignant change of the lip caused by long-term sun exposure and possibly aggravated by the use of tobacco products. Clinically it consists of a whitened, thin epithelium with or without fissures and slow-to-heal erosions. It may look and feel like "chapping," but it is found almost exclusively on the lower lip, which receives more ultraviolet exposure than the upper lip because it protrudes forward and is oriented upward. This parallels the 10:1 increased incidence of an invasive malignant lesion on the lower lip compared with the upper lip.

The carbon dioxide laser is well suited to precisely remove this premalignant epithelium with less morbidity and lower recurrence rates than other available techniques, such as liquid nitrogen, topical fluorouracil, tretinoin (Retin-A), and vermilionectomy with mucosal advancement flap repair.

The vermilion border is marked and the entire vermilion infiltrated with local anesthetic to smooth its surface, which may be preceded by a submental block or topical mucosal anesthetic to ease the pain of injection. The carbon dioxide